Public Health Subcommittee
Maine Air Toxics Advisory Committee

Recommendations for the Toxicity Factor for Diesel Particulate Matter

Revision of June 23, 2004

1.0 Introduction

The Public Health Subcommittee of the Maine Air Toxics Advisory Committee (ATAC) was charged with providing the ATAC with recommendations as to how diesel particulate matter (DPM) should be ranked within the overall Air Toxics Priority List (ATPL). This charge stemmed from the ATAC's desire to understand the basis for DEP's initial qualitative ranking of DPM in the Strawman Priority List. In order to address the charge, the Subcommittee conducted closer evaluation of the available emissions information, toxicological data, estimates of health risks from DPM exposure, and the available information on potential levels of DPM in Maine.

This memo conveys the recommendations of the Subcommittee with respect to ranking DPM and outlines the relevant information that lead to the recommendations, including other options for ranking that the Subcommittee considered.

2.0 Recommendations of the Subcommittee

The Public Health Subcommittee of the Air Toxics Advisory Committee recommends that diesel particulate matter be ranked using a range of toxicity weights from 360 to 2100. The lower toxicity weight (360) is based on a USEPA Reference Concentration and is considered protective for the potential noncancer effects of chronic exposure to DPM. The higher toxicity weight (2100) reflects an estimate of the inhalation unit risk for DPM developed by California's Environmental Protection Agency (CA-EPA). The Subcommittee acknowledges that the scientific community does not appear to have reached a consensus on an inhalation cancer unit risk for DPM. USEPA elected not to develop a quantitative estimate of cancer risks from inhalation of DPM due to the uncertainty in the available data. However, the Subcommittee felt that neglecting the potential carcinogenicity of DPM in the ranking process was not acceptable given the general consensus that DPM exposure is associated with an increased risk of cancer.

The Subcommittee further recommends that, in ranking DPM within the ATPL, DEP take into consideration the range of potential ranks (calculated using both of the toxicity weights above) as well as qualitative information on the acute and chronic noncancer effects and carcinogenicity of DPM.

The Subcommittee recognizes that several other constituents of diesel exhaust have been ranked independently in the ATPL (Ammonia, Biphenyl, Dioxins and Furans, Ethyl Benzene, Hexane, Mercury, MTBE, Naphthalene, Propionaldehyde, Styrene, Toluene, and Xylenes). Health effects that are associated with diesel exhaust may be attributable to DPM, to other constituents, or to the mixture as a whole. The toxicity of diesel

exhaust as a whole may be overestimated by ranking DPM in addition to some of the other constituents, especially where the cancer endpoint is concerned. The critical study upon which the DPM unit risk was based involved humans exposed to diesel exhaust as a whole, not just the DPM component. The Subcommittee acknowledges this issue and suggests that, as the ATAC considers the prioritization of mitigation efforts, a more integrated approach to diesel exhaust as a whole should be applied.

3.0 Overview of Ranking within Air Toxics Priority List

In the MATI process, ranking emissions of toxic air contaminants for Maine involves ranking by the product of the total emissions of each toxicant (in pounds) by a unitless toxicity weight. The toxicity weight is based on USEPA's standard quantitative values relating dose to health response[1]. Further detail on the toxicity weight is available in the Revised Draft Prioritized List of Air Toxics for the State of Maine & Basis Statement.

In order for a chemical (or chemical group) to be numerically ranked in this scheme, there must be a quantitative estimate of the chemical's in-state emissions and a toxicity weight applicable to the chemical. Absent either of these values, the chemical cannot be quantitatively ranked within the ATPL. This does not imply that the chemical cannot be given priority in the Maine Air Toxics Initiative, as qualitative information can be brought to bear for purposes of ranking.

4.0 Overview of DPM Mixture

Diesel exhaust is a complex mixture of chemical constituents existing in either gas or fine particulate form. Because diesel exhaust results from the combustion of diesel fuel, its composition can vary depending upon engine type, operating conditions, and fuel composition. Gaseous constituents of diesel exhaust may include carbon monoxide, carbon dioxide, nitrogen compounds, sulfur compounds, and low molecular-weight hydrocarbons. Toxicologically significant compounds emitted in diesel exhaust include formaldehyde, acrolein, benzene, 1,3-butadiene, polycyclic aromatic hydrocarbons (PAHs)[2], and nitro-substituted PAHs.

The particulate fraction of diesel exhaust (diesel particulate matter, or DPM) is often used as a surrogate for exposure to diesel exhaust. DPM particles are very small. According to the Tenth Report on Carcinogens (NTP, 2002), approximately 98% of DPM particles are less than 10 μm in diameter, 94% of DPM particles are less than 2.5 μm, and 92% are less than 1 μm in diameter. The small size of DPM particles has important implications

[1] For the Risk-Screening Environmental Indicators model, USEPA devised a system of unitless toxicity weights calculated from the following equations:

Toxicity weight = 0.5 / Reference Dose (in mg/kg-d)
Toxicity weight = 1.8 / Reference Concentration (in mg/m3)
Toxicity weight = Slope Factor (in risk per mg/kg-d) / 0.0005
Toxicity weight = Unit Risk (in risk per mg/m3) / 0.00014

DEP also used these equations to calculate toxicity weights for use in ranking in the Air Toxics Priority List.

[2] USEPA estimates that PAH and PAH derivatives represent only about 1% of DPM (USEPA 2002).

for the toxicity of DPM; particles of this size are highly respirable and can be inhaled deep into the lung. In addition, the small particle size results in large surface area to which organic compounds may adsorb.

Because of its size, DPM is a component of ambient particulate matter characterized as $PM_{2.5}$ (particulate matter less than 2.5 μm in diameter)[3]. USEPA estimates that, on a nationwide basis, DPM represents about 6% of total ambient $PM_{2.5}$ concentrations. Various estimates of DPM as a fraction of $PM_{2.5}$ have been made using different estimation methods. The results range from a low of 3% (in Rochester, NY) to a high of 36% (for urban Los Angeles, CA). Estimates for Manhattan in NY, NY are even higher. In general, the fraction is higher for urban areas, consistent with the greater density of diesel-burning engines in urban areas.

5.0 Available DPM Emissions Information or Estimates

Fine Particulate Matter ($PM_{2.5}$) from a variety of sources is inventoried by Maine DEP and USEPA. To compile an inventory of Diesel $PM_{2.5}$ for the Air Toxics Priority List (ATPL) inventory, the MEDEP used the 2002 preliminary National Emissions Inventory (NEI). MEDEP recommends this inventory since the preliminary 2002 NEI is the most recent inventory covering Maine, the inventory methodology is well documented, the data are generated using peer reviewed models, has undergone an EPA quality assurance review, and the information is available on the web. The disadvantage is that the inventory does not use some state specific inputs, and has not undergone a full quality assurance review by MEDEP. MEDEP also considered using the final 1999 NEI. The advantages of the 1999 NEI data is that it has undergone a full quality assurance check by both MEDEP and USEPA, and was developed based on state specific emissions assumptions, rather than national factors. However, since the 1999 data is over four years old, and vehicle miles traveled vary fairly considerably from year to year, MEDEP believes that the preliminary 2002 inventory is more accurate. Further, by using peer-reviewed models and undergoing internal quality assurance checks, the information in the preliminary 2002 NEI will be reliable. Therefore, MEDEP used the preliminary 2002 NEI inventory as a basis for the diesel $PM_{2.5}$ inventory that was used to develop the draft Air Toxics Priority List. From the preliminary 2002 NEI, MEDEP extracted the $PM_{2.5}$ emissions data that was attributed to mobile sources, both on-road and off-road, that burned diesel fuel. More information on the source of this data is available on EPA's Air Chief Website at: **http://www.epa.gov/ttn/chief/net/2002inventory.html**.

6.0 Toxicological Data

Available data suggest that diesel exhaust is associated with noncancer health effects related to both acute and chronic exposure, and that diesel exhaust is a likely carcinogenic. USEPA (2002) noted that both human and animal toxicological data suggest that acute exposure to diesel exhaust can cause irritation of the eye, throat, and bronchi, neurophysiological symptoms such as lightheadedness and nausea, and

[3] PM2.5 is regulated as a criteria pollutant, and the current standard for annual average concentration is 15 ug/m3.

respiratory symptoms such as coughing and phlegm production. The respiratory effects of chronic diesel exhaust exposure include inflammation and histopathological changes (USEPA, 2002). Finally, several groups have concluded that diesel exhaust is likely to be a human carcinogen. The National Institute for Occupational Safety and Health (NIOSH) recommended that diesel exhaust be considered an occupational carcinogen in 1988. IARC (1989, as cited in CA-EPA, 1998) classified diesel exhaust in Group 2A, reflecting sufficient animal evidence and limited human evidence of carcinogenicity. In 1990, the state of California classified diesel exhaust as a chemical known to cause cancer under Proposition 65.

In the 2002 Health Assessment Document for Diesel Engine Exhaust, USEPA characterized diesel exhaust as "likely to be carcinogenic to humans by inhalation". USEPA noted that the evidence for a carcinogenic effect in humans from occupational studies strongly supports a finding of carcinogenicity, although there is uncertainty in the treatment of confounding factors as well as uncertainty in the measures of diesel exhaust exposure in some of the epidemiological data. Further, studies of long-term, high-dose inhalation exposure to rats have shown a strong lung cancer response. USEPA and several other groups have cautioned that the mechanism of carcinogenic action that appears to be at work in rats may not have applicability in humans. These groups postulate that the rat lung cancer response results from diesel particles overloading the lung and impairing lung clearance mechanisms, thereby causing an inflammatory response. The inflammation is suggested to lead to cellular proliferation, increasing the probability of a carcinogenic mutation. Since particle overload conditions are not expected to occur in humans exposed to diesel exhaust at environmental levels, the relevance of the rat data to humans is in question.

6.1 DPM Reference Concentration. In contrast to the animal data for carcinogenic effects, the animal data for noncancer respiratory effects are generally deemed appropriate for extrapolation to human effects. USEPA used a study of rats to derive a reference concentration (RfC) of 5 µg/m3 for DPM (as a surrogate for diesel exhaust). CA-EPA (1998) concurred with USEPA's use of the rat data, and adopted the RfC of 5 µg/m3 for DPM. The critical toxicological effects observed in the rat study are pulmonary effects, histopathology, and inflammatory respiratory effects. Recent studies show that inflammatory effects may occur in humans at equivalent or lower levels of DPM than those that caused effects in rats. If these data were considered in a revision to the RfC, the resulting RfC would likely be lower[4]. If the RfC were lowered, then the noncancer toxicity weight used in the ATPL (based on the current RfC) would underestimate the actual noncancer toxicity of DPM.

6.2 DPM Inhalation Unit Risk. Epidemiological data on the carcinogenicity of inhaled DPM in a variety of occupational settings (railroad workers, mine workers, bus garage

[4] USEPA used a cumulative uncertainty factor of 3 to extrapolate from effects in rats to effects in humans, because USEPA concluded that rats might be more sensitive to DPM effects than humans. If the more typical uncertainty factor of 10 were used to account for interspecies extrapolation, the RfC would drop to 1 or 2 ug/m3.

workers, trucking company workers, etc.) are available. USEPA (2002) concluded that the human data on DPM carcinogenicity were "too uncertain" to derive a quantitative estimate of the cancer unit risk, and that the rat data were not appropriate for extrapolation to human carcinogenicity at environmental levels of DPM exposure. As a result, USEPA elected not to develop a unit risk for DPM. Notwithstanding the uncertainties in the human data, CA-EPA (2001) reported a range of unit risk estimates (using different data sets and a variety of modeling approaches) from 1.3×10^{-4} to 1.5×10^{-3} per µg/m3 DPM from analyses of two studies of U.S. railroad workers (Garshick et al. 1987 and 1988, as cited in CA-EPA, 1998). California's Scientific Review Panel suggested a value of 3×10^{-4} as the "reasonable estimate" of unit risk. CA-EPA's evaluation of the railroad worker epidemiological data has been the subject of some controversy. Alternative analyses of the data have failed to show a dose-response relationship, while additional analyses by CA-EPA staff continue to find such evidence (see **www.oehha.ca.gov** for more information). At this time, there does not appear to be a scientific consensus as to the best method for analyzing the data, nor on the appropriateness of these data for quantitative dose-response assessment.

7.0 Calculation of Toxicity Weights Using Available Toxicity Data

Using the RSEI equations for calculating toxicity weights and available toxicity values, one can calculate both noncancer and cancer toxicity weights for DPM. For noncancer endpoints, using the RfC of 5 µg/m3, one can calculate a toxicity weight of 360 for DPM. Using the CA-EPA "reasonable" unit risk of 3×10^{-4} per µg/m3, one can calculate a toxicity weight of 2100 for DPM. The range of unit risks calculated by CA-EPA would correspond to a range of toxicity weights from 930 to 11,000.

It is important to recognize that none of the available toxicity values provides a mechanism for considering the acute effects of DPM exposure. In fact, health effects resulting from acute exposures are not considered at all in the current ATPL ranking scheme.

8.0 CalEPA Prioritization of Toxic Air Contaminants under Children's Environmental Health Protection Act: Rationale for Ranking DPM in Tier 1

Under a mandate from California's Children's Environmental Health Protection Act, CA-EPA conducted an evaluation of the unique susceptibility of children to the health effects of DPM. The goal of the evaluation was to determine whether DPM should be ranked within the top tier (Tier 1) of toxic air contaminants that could cause infants or children to be susceptible to illness. CA-EPA concluded that DPM should be ranked in Tier 1 based on both greater exposures to children and on evidence of greater toxicological susceptibility. The major findings of CA-EPA's review are as follows:

- There is evidence to suggest that DPM may facilitate the development of allergies and may potentiate allergic responses in susceptible individuals. In addition, there is mechanistic support for the role of DPM in childhood asthma.

- A number of epidemiological studies have related truck traffic density with respiratory symptoms in children.
- DPM contributes to ambient particulate matter (particularly $PM_{2.5}$). Particulate matter has been shown to be associated with bronchitis, coughing, wheezing, and exacerbation of asthma.
- Diesel exhaust contains PAHs, which are associated with immunosuppressive effects, genotoxicity, and low birth weight. Further, diesel exhaust contains a variety of genotoxic agents, and some studies have shown higher cancer risks associated with early-life exposure to genotoxic agents than similar exposures later in life.
- Children's exposure to DPM may be greater than adults. Specifically, children experience higher particle doses per lung surface area than similarly exposed adults. In addition, children's oral exposures to settled DPM particles are also higher because of hand-to-mouth activity.

9.0 Estimates of Ambient DPM Concentrations and Corresponding Risks

9.1 NATA Estimates of DPM Concentrations. For the National Air Toxics Assessment (NATA), USEPA estimated concentrations of DPM in Maine but did not calculate risks. The highest county-specific median exposure concentration of DPM in Maine was in the range from 0.45 to 0.7 µg/m3. These levels are well below the RfC of 5 µg/m3. Using the CA unit risk of 3×10^{-4} per µg/m3, these exposure concentrations would correspond to estimated cancer risks in the range of 1 or 2 in 10,000.

9.2 to Estimates of DPM Concentrations from Local PM2.5 Data. Although Maine does not have data on ambient levels of DPM, monitoring data on $PM_{2.5}$ can be used with estimates of the fraction attributable to DPM[5] to generate crude estimates of the range of potential DPM concentrations in Maine. DEP[6] reports that the highest annual average $PM_{2.5}$ concentrations from urban monitoring locations in Maine[7] are in the range of 11-12 µg/m3. These levels have been observed in locations such as Madawaska, Lewiston/Auburn, and Portland (near Tukeys Bridge, considered a worst-case location).

In 1995, the Northeast States for Coordinated Air Use Management conducted a study of fine and coarse particle concentrations and chemical compositions in the northeastern states (Salmon et al., 1999)[8]. Particle concentrations and compositions were measured in 24-hour samples every 6 days over the course of the year. Three sites in Massachusetts (Kenmore Square in Boston, suburban Reading, and the Quabbin Reservoir) and 2 sites in New York (downtown Rochester and rural Brockport) were selected for sampling. Salmon et al. reported annual average fine particle concentrations (less than 2.2 µm in

[5] USEPA estimates that about 94% of DPM is smaller than 2.5 um in diameter.
[6] Personal communication, Jeff Emery, DEP.
[7] DEP reports that PM 2.5 levels in rural areas are in the range of 6-8 ug/m3.
[8] This study was used in the 2002 EPA Health Assessment Document for Diesel; however, the EPA report cites a 1997 progress report, while the data reported herein are from the 1999 final report. There are discrepancies between EPA's summary of the Boston data and the data from the final version used here; it is not clear why these discrepancies exist.

diameter) and elemental carbon fractions for all five sites. Using these data with EPA's suggested formulas for calculating the range of DPM concentrations based on elemental carbon (EC) measurements, one can calculate the ranges of DPM concentrations and the ranges of DPM as a percent of $PM_{2.2}$ for these locales. Table 1 shows the estimates of DPM concentration and DPM as a percent of $PM_{2.2}$ [9]. These data suggest DPM concentrations ranging from 0.2 to 1 ug/m3 and DPM as a percent of $PM_{2.2}$ ranging from 2% to about 9%. Excluding the data from Boston, where the contribution of elemental carbon was much higher than in other locales (likely due to the high degree of urbanization in the sampled location), the upper bound estimate of the percent of $PM_{2.2}$ attributable to DPM drops to 7%.

Table 1. Estimates of DPM as Percent $PM_{2.2}$ from NESCAUM Study

					DPM Conc. (ug/m3)*			DPM % of $PM_{2.2}$		
Location	**Location Type**	**$PM_{2.2}$ Conc. (ug/m3)**	**EC % Total**	**Conc. EC (ug/m3)**	**Low**	**Point**	**High**	**Low**	**Point**	**High**
Kenmore Square, Boston, MA	Urban	16.2	7%	1	0.7	1	1	4%	6%	9%
Reading, MA	Suburban	14.6	5%	0.7	0.5	0.6	1	3%	4%	7%
Quabbin Reservoir, MA	Rural/ Background	12.4	3%	0.4	0.2	0.3	0.5	2%	3%	4%
Rochester, NY	Urban	14.9	4%	0.6	0.4	0.5	0.8	2%	4%	5%
Brockport, NY	Rural/ Background	12.8	3%	0.4	0.2	0.3	0.5	2%	3%	4%

*EPA (2002) suggests these formulae for calculating DPM from EC: low DPM = EC*0.62, point estimate DPM = EC*0.89, and high DPM = EC*1.31.

Using the range of DPM as a percent of PM2.5 from the NESCAUM study, with DEP's high-end PM2.5 concentrations (11-12 ug/m3), one can calculate a range of DPM concentration estimates from 0.2 to 0.8 ug/m3 in the areas where higher PM2.5 concentrations have been measured. These estimates compare favorably with the NATA model predictions of DPM concentrations in Maine (median exposure concentrations from 0.45 to 0.7 ug/m3). If these estimated levels are compared with the RfC of 5 µg/m3, the risk of noncancer effects at ambient concentrations of DPM appears to be low. Using the CA-EPA unit risk of 3×10^{-4} per µg/m3, one can estimate potential cancer risks ranging from 6 in 100,000 to about 2 in 10,000 for persons living in areas with the highest $PM_{2.5}$ measurements, although the substantial uncertainty associated with these risk estimates cannot be overstated. These concentrations and risk estimates are based on 1995 data on DPM as a fraction of fine particles from other locations within the northeast. Differences in meteorological conditions and degree of urbanization between the measured sites and sites in Maine, in addition to changes in diesel emissions

[9] Measurements of $PM_{2.2}$ will slightly underestimate the $PM_{2.5}$ concentration by excluding particles between 2.2 and 2.5 µm in diameter. However, only about 2% of diesel particles are in the 1.0 to 2.5 µm range; 92% are below 1.0 µm in diameter.

since 1995, could contribute to the uncertainty. These risk estimates are useful only from the perspective of comparing DPM with other air toxics in Maine.

In its Health Assessment Document for Diesel Engine Exhaust, USEPA conducted an "exploratory risk analysis" which estimated that cancer risks associated with environmental levels of DPM exposure nationally may exceed 1 in 100,000 and may be as high as 1 in 1,000. However, the agency acknowledged that the risk could be lower or even zero.

10.0 Expected Impact of Pending Regulations Affecting Diesel Engines and Emissions

Beginning with 2007 model year, diesel vehicles will have to meet new emissions standards, and USEPA is requiring that diesel fuel be produced with much lower sulfur content. The availability of lower sulfur fuel may result in immediate reductions in particulate matter from existing vehicles, although the degree of reduction is not known. Reductions associated with changes in emissions standards will occur more slowly, as the fleet of diesel vehicles in use is gradually replaced with newer vehicles meeting the standards.

The agency conducted a Regulatory Impact Analysis of this rule in 2000. This analysis suggested that annual average ambient $PM_{2.5}$ concentrations (averaged across the U.S.) in the year 2030 would be lower by about 3% (or 0.27 µg/m3) as a result of this rule. The effect on air quality in urban areas would be greater; the agency estimated population-weighted average concentrations to be lower by about 4% or 0.65 µg/m3 (USEPA, 2000). It is important to remember that the effect on $PM_{2.5}$ will depend on the contribution of DPM to $PM_{2.5}$ in any given area, and that urban areas will experience the greatest reductions.

11.0 Options Considered for Ranking DPM:

Several options were considered for ranking DPM within the Air Toxics Priority List, including:

1. *Implement research to estimate diesel contribution to measured PM levels in Maine.* There is no quantitative information on the contribution of DPM to PM levels in Maine. Sampling aimed at identifying the contribution of DPM to PM has been undertaken elsewhere in the country, but the relevance of these estimates to DPM levels in Maine is uncertain. A research program could be undertaken to fill this data gap. This effort could take a year or more, given the need to estimate annual average concentrations. In addition, the costs associated with analysis of PM measurements for elemental carbon (if this method for estimating DPM were chosen) would be a factor. Finally, because of the uncertainty in the quantitative toxicological data, further refinements of the estimates of DPM concentration may not significantly improve risk estimates.

2. *Implement quantitative ranking of DPM using toxicity weights based on available toxicity values, however uncertain*. Quantitative ranking of DPM using the current ATPL index depends heavily on the availability of estimates of DPM emissions. Toxicity weights based on the RfC and CA unit risks could be used if emissions information were available, provided that the uncertainty in the toxicity weights is documented and considered in a qualitative fashion.
3. *Use qualitative ranking based on toxicological information documenting effects on children, carcinogenicity, respiratory effects, as well as available semi-quantitative risk estimates.* In the absence of emissions information, a qualitative ranking approach could be used based on the information assembled in this report. It is important, however, that any use of the risk estimates in this report acknowledge the substantial uncertainty in the risk estimates due to uncertainty in both the unit risk and in estimates of ambient DPM concentrations.

12.0 References

CA-EPA, 1998. Part B: Health Risk Assessment for Diesel Exhaust. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment.

CA-EPA, 2001. Prioritization of Toxic Air Contaminants – Children's Environmental Health Protection Act: Diesel Particulate Matter.

Maine Air Toxics Advisory Committee, Revised Draft Prioritized List of Air Toxics For the State of Maine & Basis Statement, May 2004 (Available from Maine Department of Environmental Protection, Augusta, ME)

NTP, 2002. Report on Carcinogens, Tenth Edition; U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, December.

USEPA. 2000. Regulatory Impact Analysis: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements. EPA420-R-00-026. December.

USEPA, 2002. Health Assessment Document for Diesel Engine Exhaust. EPA/600/8-90/057F. May.